Personally Modifiable Risk Factors Associated with Pediatric Hearing Loss: A Systematic Review
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Otolaryngology -- Head and Neck Surgery 2014 151: 14 originally published online 26 March 2014
DOI: 10.1177/0194599814526560
The online version of this article can be found at:
http://oto.sagepub.com/content/151/1/14

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What is This?
Personally Modifiable Risk Factors Associated with Pediatric Hearing Loss: A Systematic Review

Adam P. Vasconcellos, MD1, Meghann E. Kyle, AuD, CCC-A1, Sapideh Gilani, MD1, and Jennifer J. Shin, MD1

Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

Abstract

Background. Pediatric hearing loss is an increasingly recognized problem with significant implications. Increasing our quantitative understanding of potentially modifiable environmental risk factors for hearing loss may form the foundation for prevention and screening programs.

Objective. To determine whether specific threshold exposure levels of personally modifiable risk factors for hearing loss have been defined, with the overarching goal of providing actionable guidance for the prevention of pediatric hearing loss.

Data Sources. A systematic review was performed. Computerized searches of PubMed, EMBASE, and the Cochrane Library were completed and supplemented with manual searches.

Review Methods. Inclusion/exclusion criteria were designed to determine specific threshold values of personally modifiable risk factors on hearing loss in the pediatric population. Searches and data extraction were performed by independent reviewers.

Results. There were 38 criterion-meeting studies, including a total of 50,651 subjects. Threshold noise exposures significantly associated with hearing loss in youth included: (1) more than 4 hours per week or more than 5 years of personal headphone usage, (2) more than 4 visits per month to a discotheque, and (3) working on a mechanized farm. Quantified tobacco levels of concern included any level of in utero smoke exposure as well as secondhand exposure sufficient to elevate serum cotinine.

Conclusions. Specific thresholds analyses are limited. Future studies would ideally focus on stratifying risk according to clearly defined levels of exposure, in order to provide actionable guidance for children and families.

Keywords

hearing loss, child, infant, adolescent, tobacco, noise, risk factor

Received November 18, 2013; revised February 6, 2014; accepted February 12, 2014.

Introduction

Pediatric hearing loss affects 1.1% to 19.5%1-3 of children and has the capacity to negatively impact language development and psychosocial maturation.3-6 Even mild or unilateral hearing loss in school-aged children may result in significantly worse quality of life, developmental delay, and compromised academic performance.7-10 In addition, childhood hearing loss may be associated with longstanding consequences, including lower graduation rates and an increased risk of unemployment.8,10,11

Pediatric hearing loss may be attributed to genetic mutations, environmental factors, or idiopathic causes.12 Among environmental exposures, some are clearly established but may occur by necessity, such as treatment with cisplatin, gentamicin, or extracorporeal membrane oxygenation.13,14 Others are challenging to prevent, such as cytomegalovirus infection, low birth weight, or meningitis.15-17 There are, however, a subset of environmental risk factors that may be more readily modified, such as noise exposure and exposure to tobacco smoke.18-22

Modifying risk factors according to specific goals has proven to minimize morbidity from multiple life-altering conditions. In primary care, for example, risk of ischemic stroke is decreased by targeting a goal blood pressure below 140/90 mmHg (with lower targets in some subgroups) via primary prevention strategies such as limiting daily sodium intake to 4 grams and encouraging 30 minutes of daily aerobic exercise.23 Likewise, risk of coronary artery disease may be mitigated by achieving a blood pressure less than 120/80, cholesterol <200 mg/dl, and eliminating smoking.24,25 These high-impact, target-specific preventive measures suggest the potential for a parallel utility with hearing loss.

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In order to apply such goal-specific prevention for hearing loss, we must first understand exactly what desired targets might be. While it has been suggested that exposures such as tobacco, heavy metals, noise, nutritional deficiencies, and low socioeconomic status are associated with pediatric sensorineural hearing loss (SNHL), data are mixed and specific targets goals are not widely accepted. Understanding the current best evidence regarding risk associated with specific exposure levels would facilitate future inquiries regarding specific preventative regimens. In addition, it may provide interim guidance in providing counseling to families that is of more utility than generalized statements such as “avoid loud noises,” in the same way that advising patients toward a blood pressure of 120/80 is more meaningful than simply stating “keep your blood pressure down.”

With these concepts in mind, we performed a systematic review with the overarching goal of evaluating potentially alterable exposures to determine what specific metrics are associated with early onset hearing loss. Systematic reviews provide a rigorous method to evaluate the current best evidence regarding a specific clinical question and are among the highest level of evidence available.26-28 Given the quantity of data involved, this systematic review is presented in two parts. This installment focuses on the risk factors that may be modified at the personal level, specifically tobacco and noise exposure. A sister article focuses on risk factors that are more likely to require societal-level changes for modification.29

Methods

Search Strategy

Computerized searches were performed to identify all relevant data. At the outset, a screening computerized PubMed search of MEDLINE (1966 to February 2013) was performed to obtain studies assessing risk factors for hearing loss in children in overview. Articles mapping to the medical subject heading hearing loss (exploded) or contained hearing loss in the title were collected into one group. Articles that mapped to the exploded medical subject heading tobacco smoke or the title, abstract, or keyword noise were collected into a second group. Articles mapping to the medical subject headings or keywords child, infant, adolescent, or pediatrics (all exploded) were collected into a third group. The 3 groups were then cross-referenced and limited to those with human subjects and English language. Studies that focused on the outcomes of cochlear implant recipients were not included. Parallel searches were performed in Embase and the Cochrane library. Among 4 independent reviewers, each search was completed in duplicate for corroboration. This search yield 4295 studies, including overlap among individual searches.

The titles of the studies obtained from these combined search methods were evaluated according to the a priori inclusion/exclusion criteria described in the following. This title review yielded 365 potential abstracts, which were reviewed in more detail according to the same criteria. Subsequently, 132 full articles were evaluated against a priori criteria in detail (Figure 1).

Inclusion and Exclusion Criteria

The articles identified by the computerized search strategy described previously were evaluated to identify those that met the following inclusion criteria: (1) study participant age entirely or predominantly 18 years or younger, (2) personally modifiable environmental risk factors for permanent hearing loss evaluated (tobacco, noise), (3) comparison between exposure to environmental risk factor and no/minimal exposure, and (4) hearing loss clearly defined (mild, moderate, severe or profound; unilateral or bilateral). Articles were excluded if (1) adults and pediatric data were combined such that pediatric data could not be reviewed distinctly; (2) age at the time of analysis was not described; (3) pediatric data were presented in a nonanalyzed subgroup as part of a primarily adult study; (4) solely nonpermanent hearing loss was assessed, such as hearing loss from acute otitis media, otitis media with effusion, or recent temporal bone fracture; or (5) hearing loss was not clearly defined, such as parent surveys to assess possible hearing impairment of their children without accompanying audiological measurements.

Data Extraction

Data extraction was focused on items relevant to the study results, potential sources of heterogeneity among those results, and study identification (author, year of publication, full reference citation). Extracted data included (1) the number of subjects in each group, (2) the number/percentage with hearing loss in each group, and (3) the P-value, confidence interval, or descriptive statistics reported. In addition, potential sources of heterogeneity among studies were included: (1) age at outcome of measurement, (2) definition of environmental risk factor assessed, (3) definition of any relevant control group, (4) audiological criteria used...
for stratification of data, and (5) study design. Independent reviewers evaluated data in duplicate using standardized tables. For studies emanating from a common database, the sample size from the study with the largest number of subjects was utilized toward the total patient count estimate, so as to avoid duplicate counts.

Quantitative Data Analysis
The primary outcome measure was risk of hearing loss in childhood and adolescence with exposure to tobacco or noise. An a priori plan was made to perform a meta-analysis if the data were appropriate, meaning that study designs and outcome measures had sufficient homogeneity. The data, however, ultimately proved too heterogeneous for a pooled analysis.

Results
Study Characteristics
These search criteria yielded 38 criterion-meeting papers with 50,651 subjects (accounting for overlapping study populations). Reports related to tobacco and noise exposure included 20 cross-sectional studies, 9 cohort studies, 3 longitudinal follow-up studies, 2 case-control studies, 2 retrospective reviews, 1 cluster-randomized controlled trial, and 1 case series. Exposure to a given risk factor was assessed by quantitative measurements in 6 studies and by self-report in 32 studies.

Impact of Tobacco Exposure on Pediatric Hearing Loss
Three cross-sectional studies and 1 cohort study assessed the risk of pediatric hearing loss with tobacco exposure. All 4 studies examined prenatal tobacco and neonatal hearing, while 1 publication addressed the effect of secondhand smoke.

Secondhand Smoke Exposure
Data from the National Health and Nutrition Examination Survey (NHANES) 2005-2006 (n = 1533) study showed a higher prevalence of unilateral and bilateral hearing loss among adolescents who had secondhand smoke exposure (identified via serum levels of cotinine, a nicotine metabolite) as compared to unexposed youth (11.82% vs 7.53%, \( P = .04 \); Table 1 and see appendix at www.otojournal.org). Cotinine levels associated with increased risk were as low as “detectable” to 0.0876 mg/L. Furthermore, when exposure was stratified into quartiles, higher serum cotinine was significantly associated with unilateral low frequency hearing loss (\( P = .02 \)).

Prenatal Smoke Exposure
Two studies evaluated the impact of prenatal smoke exposure on hearing in adolescence (Table 2 and see appendix at www.otojournal.org). One study demonstrated a trend toward a higher risk of unilateral low frequency hearing loss (mean 0.5, 1, 2 kHz) with prenatal smoke exposure in NHANES participants 12 to 15 years of age (16.8% vs 6.4%, \( P = .11 \)). The NHANES 2005-2006 data set was also evaluated in a more recent cross-sectional study, which demonstrated significantly worse pure tone hearing in adolescents with a history of prenatal smoke exposure (worse hearing ear at 2 and 6 kHz, right/left mean at 6 kHz). Both studies defined sensorineural hearing loss as pure tone thresholds >15 dB with normal otoscopy and tympanometry, but the former evaluated multiple frequencies grouped together, while the latter analyzed single frequencies in isolation.
Two neonatal analyses also demonstrated a significant impact of prenatal smoke on cochlear function as measured by transient evoked otoacoustic emissions (TEOAE). One cross-sectional study of 418 neonates showed a statistically significant negative impact of smoking during pregnancy on TEOAE, with even the lowest exposure level (1 to 4 cigarettes per day) associated with worse hearing than with no exposure ($P < .001$). There was not an overt dose-dependent response, as there were no significant differences among the low, medium, and high exposure groups. The second study (n = 200 neonates) also demonstrated a lower TEOAE mean response overall and at 4000 Hz specifically ($P = .001$). This difference was observed in even the lowest exposure group of less than 5 cigarettes daily ($P = .01$) and persisted as the intermediate and higher dose groups were compared to the nonsmoking group ($P = .01$, .04). Again, increasing the dose of tobacco exposure did not further increase the impact on hearing among those who were exposed.

### Impact of Noise Exposure on Pediatric Hearing Loss

Sixteen cross-sectional studies, 9 cohort studies, 3 longitudinal follow-up studies, 2 case-control studies, 2 retrospective reviews, 1 cluster-randomized control trial, and 1 case series assessed the impact of noise exposure on pediatric hearing loss.

#### Personal Listening Devices

Two studies demonstrated a significantly increased risk of hearing loss with personal headphone usage and specified a threshold of concern. One large cross-sectional study of adolescents (n = 1294) suggested that more than 4 hours per week of “loud music through headphones” was associated with a significantly increased risk of hearing loss (prevalence 22.5% vs 15%, $P = .03$). A second, smaller, cross-sectional study (n = 490) showed significantly elevated hearing thresholds in adolescents who reported using portable music players for >5 years, as compared with students with no portable music usage. 

One cross-sectional analysis (n = 177) attempted to quantify an at-risk volume of personal listening devices. Each subject’s preferred listening volume was evaluated and translated into an associated 8-hour equivalent continuous sound level; there was a positive correlation between total sound exposure and elevated hearing thresholds at 2 extended high frequencies (11.2 and 14 kHz), but a threshold value for concern was not reported. Of note, this study also found that subjects who tended to listen to louder music also listened for longer duration ($P = .012$).

Two cross-sectional studies concluded that although there was a significant association between headphone usage and hearing loss, the specific number of hours with a personal listening device did not make a definitive difference. The first study evaluated adolescent females of low socioeconomic status (n = 2698, mean age 15.8 years) and demonstrated a significant relationship between daily personal stereo usage and sensorineural loss (PTA 16-40dB at low or high frequencies). The number of hours of listening, however, was not different between the SNHL and normal hearing groups.

One cohort study (mean age 15.8 years) and 2 longitudinal studies found no significant correlation between frequent personal stereo use and hearing acuity.

The specific details of the aforementioned studies are presented in Table 3 and the appendix at www.otojournal.org.

### Concerts, Discotheques, and Ambient Loud Music

Four cross-sectional studies, 2 longitudinal studies, and 1 cohort study explored associations between hearing loss and visits to loud environments, such as discotheques and concerts, with 1 study delineating a threshold value of concern. In a cross-sectional study of 14- to 15-year-old students (n = 1294), those with 4 or more visits per month had a significantly higher risk of hearing loss, as compared to those with less exposure (24.5% vs 15.0%, $P = .027$). Visits exceeding 4 hours duration trended toward worse hearing, but were not significantly different. A longitudinal follow-up study (n = 106 adolescents) stratified risk with a more
generalized measure of exposure frequency and found that those with hearing loss were significantly more likely to self-report “medium” as opposed to “rare” or “no” total music exposure \((P < .001, \text{ girls and boys})\), “medium” to “frequent” live concert attendance \((P < .06, \text{ boys})\), “frequent” attendance at discos \((P < .009, \text{ girls})\), and “high” exposure to music at home \((P < .012, \text{ girls})\).²¹

Three other studies demonstrated a significant association between concert/discotheque noise exposure and pediatric hearing loss but did not report specifically delineated levels of exposure. One cross-sectional study \((n = 214, 14-19\) years old) demonstrated a statistically significant association between concert attendance and hearing loss.²⁰ A small prospective cohort \((n = 14)\) demonstrated significant high frequency hearing loss after repeated controlled exposures to live rock-and-roll music, but also noted that hearing thresholds had improved at the 5-month follow-up.²¹ Another longitudinal study demonstrated significant hearing changes correlated with frequent discotheque attendance over a 12-week period.²⁹

Table 2. Impact of Prenatal Tobacco Exposure on Pediatric Hearing Loss.

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Study Design (sample size)</th>
<th>Age</th>
<th>Tobacco Exposure Measure</th>
<th>Hearing Evaluation</th>
<th>Follow-up Time</th>
<th>Results/Conclusions</th>
<th>Additional Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Korres, 2007²²</td>
<td>Cohort of exposed and unexposed neonates ((n = 200))</td>
<td>Newborns (24-48 hours after birth)</td>
<td>Maternal smoking during pregnancy (parental report)</td>
<td>Click-evoked otoacoustic emissions (TEOAEs)</td>
<td>N/A</td>
<td>Prenatal exposure to tobacco smoking results in lower mean response TEOAEs and mean amplitudes at 4000 Hz.</td>
<td>Differences between smoking and nonsmoking mothers analyzed using (t) test for unpaired data</td>
</tr>
<tr>
<td>Weitzman, 2013³¹</td>
<td>Cross-sectional ((n = 964))</td>
<td>12-15 yrs (\text{NHANES (2005-2006)})</td>
<td>Maternal smoking during pregnancy (parental report)</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>Across all frequencies, the mean pure tone hearing level was worse in those with prenatal smoke exposure than in those who were unexposed, with statistically significant elevations noted at 6 kHz for mean of right/ left ears and at 2 and 6 kHz for the worse hearing ear.</td>
<td>(t) test, multivariate odds ratio</td>
</tr>
<tr>
<td>Lalwani, 2011⁹</td>
<td>Cross-sectional ((n = 832))</td>
<td>12-15 yrs (\text{NHANES (2005-2006)})</td>
<td>Maternal smoking during pregnancy (parental report)</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>There was a trend toward higher rates of SNHL with prenatal smoking exposure, but the results were not statistically significant.</td>
<td>Chi-square test</td>
</tr>
<tr>
<td>Durante, 2011³⁰</td>
<td>Cross-sectional ((n = 418))</td>
<td>Neonates</td>
<td>Maternal smoking during pregnancy (parental report)</td>
<td>Click-evoked otoacoustic emissions (TEOAEs)</td>
<td>N/A</td>
<td>Maternal smoking during pregnancy was associated with a negative impact on TEOAE results. The lowest and highest level of cigarettes smoked resulted in a significant worsening as compared to control. There was no significant difference between exposure levels.</td>
<td>Analysis of variance (ANOVA)</td>
</tr>
</tbody>
</table>

Abbreviations: TEOAE, transient evoked otoacoustic emissions; NHANES, National Health and Nutrition Examination Survey; PTA, pure tone audiometry; SNHL, sensorineural hearing loss.
<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Study Design</th>
<th>Age</th>
<th>Hearing Evaluation</th>
<th>Follow-up Time</th>
<th>Results/Conclusions</th>
<th>Additional Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cone, 2010³⁷</td>
<td>Cross-sectional (n = 5278)</td>
<td>7-12 yrs (mean)</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>Personal stereo use (parental report) was found to have a statistically significant association with slight-mild SNHL.</td>
<td>Adjusted OR, 95% CI for grade, sex, and school type</td>
</tr>
<tr>
<td>Berg, 2011³⁶</td>
<td>Cross-sectional (n = 2698)</td>
<td>12-20 yrs</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>Significant relationship between regular personal listening device use and high frequency hearing loss in adolescent low socioeconomic status females.</td>
<td>Female adolescents of a residential foster care facility in a metropolitan area</td>
</tr>
<tr>
<td>Niskar, 1998²</td>
<td>Cross-sectional (n = 6166) NHANES (1988-1994)</td>
<td>6-19 yrs</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>Use of headphones or exposure to loud noise within 24 hours of hearing assessment did not appear to affect the prevalence of measured LFHL or HFHL.</td>
<td>Prevalence estimates and 95% confidence intervals</td>
</tr>
<tr>
<td>Henderson, 2011¹⁹</td>
<td>Cross-sectional (n = 2519: NHANES 1988-1994; n = 1791: NHANES 2005-2006)</td>
<td>12-19 yrs</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>No significant relationship observed between loud music or personal listening device use with headphones within 24 hours of PTA measurement.</td>
<td>Multivariate noise induced threshold shift (NITS) odds ratio, adjusted for age, gender, race, PIR</td>
</tr>
<tr>
<td>Weichbold, 2012²³</td>
<td>Cross-sectional (n = 1294)</td>
<td>14-15 yrs</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>Statistically significant association between time listening to loud music with headphones and hearing loss in adolescents, driven by high-exposure risk group (&gt;4 hours in a given week).</td>
<td>Chi-square test</td>
</tr>
<tr>
<td>Kim, 2009³⁴</td>
<td>Cross-sectional (n = 490)</td>
<td>13-18 yrs</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>Significant worse hearing in adolescents with personal music players usage &gt;5 years, compared with students not using them.</td>
<td>t test</td>
</tr>
<tr>
<td>de Beer, 2003³⁸</td>
<td>Cohort (n = 238)</td>
<td>12-20 yrs</td>
<td>Audiometric threshold PTA</td>
<td>Follow-up of birth cohort at 8 yrs, hearing assessed at 18</td>
<td>No significant correlation was found between frequent personal stereo exposure and hearing acuity.</td>
<td>Highest and lowest tertiles of personal stereo exposure selected for statistical analysis</td>
</tr>
<tr>
<td>Sulaiman, 2013³⁵</td>
<td>Cross-sectional (n = 177)</td>
<td>13-16 yrs</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>Subjects' sound exposure levels from the devices were positively correlated with their hearing thresholds at 2 of the extended frequencies (11.2 and 14 kHz)</td>
<td>-8-hour equivalent continuous sound level calculated -Pearson's correlation -Chi-square test</td>
</tr>
<tr>
<td>Bassoni, 2005³¹</td>
<td>Longitudinal follow-up study (n = 102)</td>
<td>14-17 yrs</td>
<td>Audiometric threshold PTA</td>
<td>4 years</td>
<td>No significant differences with regard to personal music player usage.</td>
<td>Categorization of frequency: no, rare, medium, frequent</td>
</tr>
<tr>
<td>Axelsson, 1993³⁹</td>
<td>Longitudinal follow-up study (n = 50)</td>
<td>5-12 yrs (approx)</td>
<td>Audiometric threshold PTA</td>
<td>12 weeks</td>
<td>No significant correlation between frequent use of personal listening devices (Walkman) and elevated hearing thresholds.</td>
<td>Average frequency and intensity of sound exposures compared</td>
</tr>
</tbody>
</table>

Abbreviations: PTA, pure tone audiometry; SNHL, sensorineural hearing loss; OR, odds ratio; CI, confidence interval; NHANES, National Health and Nutrition Examination Survey; LFHL, low frequency hearing loss; HFHL, high frequency hearing loss; PIR, poverty-income ratio.
One cross-sectional study (n = 872) found no significant association between amplified music exposure and hearing loss (odds ratio [OR], 0.81; 95% confidence interval [CI], 0.57-1.15), but did find a difference associated with other noise exposure.\(^2\) A second cross-sectional study (n = 500) observed no statistically significant correlation between attending >10 pop rock concerts and hearing thresholds among military conscripts.\(^4\) Additional specific details of these studies are presented in Table 4 and the appendix at www.otojournal.org.

**Farms, Airports, School Workshops, and Urban Environments**

Farm youth were evaluated in 2 studies that both demonstrated a significant impact on hearing. One cross-sectional study showed that 12 to 19 year olds (n = 872) actively involved in work on a modern mechanized farm had an increased prevalence of mild hearing loss and early noise-induced hearing loss.\(^4\) An observational cohort compared children of families living on a farm (n = 204, ages 6-19) with adolescents in NHANES 1988-1994 and demonstrated significantly worse hearing (high frequency and any type) in the farm youth.\(^4\)

One retrospective cohort study and one case-control study both suggested that living or attending school close to an airport was associated with pediatric hearing loss.\(^4,45\) The cohort study (n = 379) demonstrated significantly worse standard pure tone average, high pure tone average, and threshold at 4 kHz in children with frequent exposure to aircraft noise (20 flights overhead daily) as compared with age-matched controls.\(^45\) The case-control study similarly suggested an association between aircraft noise exposure and high frequency hearing loss in youth, but findings were not statistically significant.\(^46\) A cross-sectional study (n = 3322) demonstrated no significant difference between the incidence of bilateral sensorineural and mixed hearing loss in elementary and high school students living near airports, as compared to population averages.\(^37\) Similarly, a retrospective cohort (n = 200) showed no significant association between hearing loss and living in neighborhoods in close proximity to an airport.\(^48\)

Noisy workshops and urban environments were evaluated in 2 reports. One cross-sectional study of adolescents (n = 214) demonstrated a statistically significant relationship between participation in noisy school workshops and hearing loss.\(^40\) One retrospective cohort (n = 194) compared auditory brainstem responses in boys who lived in urban and rural environments and demonstrated no significant differences in peak or interpeak latencies.\(^49\) Additional specific details of these studies are presented in Table 5 and the appendix at www.otojournal.org.

**Impulse Noises**

Four studies demonstrated an association between sensorineural hearing loss and exposure to impulse noises, such as firearms and firecrackers. One study quantified exposure via time spent in the military; youth (n = 39) had significantly worse median thresholds, particularly in the right ear, after exposure. A retrospective cohort study quantified frequency and duration of firearm exposure with respect to hearing loss and demonstrated that 17.6% (12/68) of adolescents had noise-induced hearing loss. Only males were affected; 11 of these subjects noted frequent gun use (≥4 times per month) and used firearms for a mean of 1.3 years longer, suggesting a potential association between gun use and noise-induced hearing loss (no statistical testing of a null hypothesis was reported; post hoc P = .087, Fisher’s exact).\(^51\)

Two studies proposed a relationship between impulse noises and hearing loss but did not quantify the quantity or duration of firearm or firecracker exposure. One retrospective chart review (n = 53) showed that the majority of children had persistent high frequency hearing loss 6 to 12 weeks after exposure to firecrackers and toy cap pistols.\(^52\) A cluster analysis of a cross-sectional study (n = 282) suggested that exposure to impulse noises such as firecrackers and explosives may potentiate a 4 kHz worsening and/or high tone deafness.\(^53\)

One cross-sectional study (n = 500) and one longitudinal study (n = 50) failed to observe a significant relationship between frequent impulse noise exposure and hearing thresholds.\(^39,43\)

The specific details of the aforementioned studies are presented in Table 6 and the appendix at www.otojournal.org.

**In Utero Exposure**

Two studies differed in their respective assessments of in utero noise exposure and early onset hearing loss (Table 7 and appendix at www.otojournal.org). A cross-sectional study (n = 131) found mothers exposed to frequent loud occupational noise during pregnancy were approximately 3 times as likely to bear children with high frequency hearing loss as compared with unexposed mothers (P < .01).\(^54\) In contrast, a case-control study found no differences in the response amplitude of distortion product otoacoustic emissions or background noise between control and study groups, suggesting that exposure to occupational noise during pregnancy does not affect the hearing of children born from exposed mothers.\(^55\) The women in this study all wore hearing protection and worked through their eighth pregnant month.

**NHANES**

Cross-sectional studies of data from the National Health and Nutrition Examination Surveys (NHANES) were described in 4 reports. The earliest (1998) evaluated NHANES III (1988-1994) and showed no association between either low or high frequency hearing loss and personal headphone use or exposure to loud noise within the prior 24 hours.\(^2\) A second study (2001) focused on NHANES III and described a noise-induced threshold shift (NITS) according
<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Study Design (sample size)</th>
<th>Age</th>
<th>Noise Exposure</th>
<th>Hearing Evaluation</th>
<th>Follow-up Time</th>
<th>Results/Conclusions</th>
<th>Additional Comments</th>
</tr>
</thead>
</table>
| Weichbold, 2012³³ | Cross-sectional (n = 1294, visit frequency; 1282, duration of stay) | 14-15 yrs | Discotheques or loud pubs: visit frequency, duration of stay                     | Audiometric threshold PTA | N/A            | The number of visits to discotheques/loud pubs was significantly associated with hearing loss in adolescents, mainly in high-exposure risk groups (4+ visits per month).  
No statistically significant association between average length of stay in discotheques/loud pubs and hearing. | Chi-square test                        |
| Axelsson, 1994⁴³  | Cross-sectional (n = 500) | 18 yrs | >10 pop-rock concerts                                                            | Audiometric threshold PTA | N/A            | No statistically significant correlation was observed between attending >10 pop-rock concerts and hearing thresholds among military conscripts.  
SNHL in teenagers was significantly related to noisy recreational activities. | Relative risks and confidence limits (95%) calculated Prevalence ratio and prevalence odds ratio |
| Martinez-Wbaldo, 2009⁴⁰ | Cross-sectional (n = 214) | 14-19 yrs | Concert attendance, school workshops                                               | Audiometric threshold PTA | N/A            | Significant high frequency threshold shifts were observed after repeated exposures to amplified live rock-and-roll music on initial follow up. At 5 months, 1 of 14 with temporary shifts had developed a persistent loss. | t test                                |
| Ulrich, 1974⁴¹    | Prospective cohort (n = 14) | 13-17 yrs | Amplified live rock-and-roll music                                               | Audiometric threshold PTA | 30 minutes after exposures, 5 months after last exposure | Adolescents with evidence of hearing loss reported significantly higher levels of total music exposure, higher exposure to music at home (girls), higher attendance at discotheques (girls), and higher live concert attendance (boys). | t test                                |
| Biassoni, 2005²¹  | Longitudinal follow-up study (n = 102) | 14-17 yrs | Discotheques attendance, live concert attendance                                 | Audiometric threshold PTA | 4 years        | Subgroup with small threshold shifts compared with subgroup with large threshold shifts at year 4 of study | Subgroup with small threshold shifts compared with subgroup with large threshold shifts at year 4 of study |
| Axelsson, 1993⁳⁹  | Longitudinal follow-up study (n = 50) | 5-12 yrs (approx) | Discotheques attendance                                                          | Audiometric threshold PTA | 12 weeks       | Significant hearing threshold shifts were correlated with frequent discotheque attendance.                | Average frequency and intensity of sound exposures compared |

Abbreviations: PTA, pure tone audiometry; SNHL, sensorineural hearing loss.
Table 5. Impact of Farms, Airports, and Urban Environments on Pediatric Hearing Loss.

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Study Design (Sample Size)</th>
<th>Age</th>
<th>Noise Exposure</th>
<th>Hearing Evaluation</th>
<th>Follow-up Time</th>
<th>Results/Conclusions</th>
<th>Additional Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thakur, 2003</td>
<td>Retrospective cohort (n = 194)</td>
<td>10-15 yrs</td>
<td>Urban environments</td>
<td>ABR</td>
<td>N/A</td>
<td>No gross differences in peak latencies were observed among ABR results comparing children living in an urban vs rural environment.</td>
<td>Unpaired t test</td>
</tr>
<tr>
<td>Berg, 2009</td>
<td>Cluster-randomized control trial (n = 690)</td>
<td>Age 15-20 yrs</td>
<td>Farm equipment exposure</td>
<td>Audiometric threshold PTA</td>
<td>3 yrs</td>
<td>Among students exposed to frequent farm noise, those exposed to a hearing conservation program reported more frequent use of hearing protection devices, but there was no evidence of reduced levels of noise-induced hearing loss.</td>
<td>-Schools were units of randomization -Use of hearing protection devices significantly higher in intervention group</td>
</tr>
<tr>
<td>Broste, 1989</td>
<td>Cross-sectional (n = 872)</td>
<td>Age 12-19 yrs</td>
<td>Farm equipment exposure</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>Teenaged schoolchildren actively involved in farmwork have a significantly increased prevalence of mild hearing loss and early noise-induced hearing loss.</td>
<td>Multivariate logistic regression adjusting for age, sex, family history, amplified music use, snowmobiles or motorcycles</td>
</tr>
<tr>
<td>Andrus, 1975</td>
<td>Cross-sectional (n = 3,322)</td>
<td>Age 6-17 yrs</td>
<td>Aircraft noise exposure</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>Incidence of bilateral sensorineural hearing loss in elementary and high school students living directly under flight paths or immediately adjacent to airport runways was not statistically different from overall averages.</td>
<td>Two tailed t test</td>
</tr>
<tr>
<td>Green, 1982</td>
<td>Case-control (n = 409)</td>
<td>Age avg: 10.2 yrs (cases) 8.1 yrs (controls)</td>
<td>Aircraft noise exposure</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>An association is suggested between aircraft noise exposure and risk of high frequency hearing loss, but not statistically significant.</td>
<td>Multiple logistic regression; unable to validate estimates of exposure from other noise sources</td>
</tr>
<tr>
<td>Chen, 1993</td>
<td>Retrospective cohort (n = 379)</td>
<td>Age 11-12 yrs</td>
<td>Aircraft noise exposure</td>
<td>Audiometric threshold PTA, ABR</td>
<td>N/A</td>
<td>PTA, high frequencies, and threshold at 4 kHz were higher in children with frequent exposure to aircraft noise as compared with age-matched controls, whereas no consistent difference was observed in ABR latencies.</td>
<td>Exposed vs unexposed school; t test</td>
</tr>
<tr>
<td>Fisch, 1981</td>
<td>Retrospective cohort (n = 200)</td>
<td>Age 3-16 yrs</td>
<td>Aircraft noise exposure</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>This study fails to demonstrate an association between exposure to aircraft noise in residential neighborhoods and hearing impairment in children.</td>
<td>Testing equipment nonuniform among groups; no reported statistical analysis</td>
</tr>
</tbody>
</table>

Abbreviations: ABR, auditory brainstem response; NHANES, National Health and Nutrition Examination Survey; OFFHHS, Ohio Farm Family Health and Hazard Study; PTA, pure tone average.
Table 6. Impact of Impulse Noises on Pediatric Hearing Loss.

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Study Design (sample size)</th>
<th>Age</th>
<th>Noise Exposure</th>
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<th>Results/Conclusions</th>
<th>Additional Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cone, 2010³⁷</td>
<td>Cross-sectional (n = 5278)</td>
<td>7-12 (mean)</td>
<td>Toys, firecrackers, referee whistles, and so on</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>Impulse noise exposure (parent report) was associated with slight-mild SNHL (nonsignificant).</td>
<td>Adjusted OR, 95% CI for grade, sex, and school type</td>
</tr>
<tr>
<td>Henderson, 2011³⁹</td>
<td>Cross-sectional (n = 1791) NHANES (2005-2006)</td>
<td>12-19 yrs</td>
<td>Firearms</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>No significant association noted between ever having used a firearm and hearing loss.</td>
<td>Multivariate NITS OR, adjusted for age, gender, race, PIR</td>
</tr>
<tr>
<td>Shargorodsky, 2010³⁶</td>
<td>Cross-sectional (n = 1769) NHANES (2005-2006)</td>
<td>12-19 yrs</td>
<td>Firearms</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>No significant association noted between ever having used a firearm and hearing loss.</td>
<td>Multivariate logistic regression odds ratio, adjusted for age, gender, race, PIR, 3+ otitis</td>
</tr>
<tr>
<td>Axelsson, 1994³³</td>
<td>Cross-sectional (n = 500)</td>
<td>18 yrs</td>
<td>Firecrackers, shooting firearms</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>No statistically significant correlation was observed between playing with firecrackers or shooting firearms and hearing thresholds among military conscripts.</td>
<td>Relative risks and confidence limits (95%) calculated</td>
</tr>
<tr>
<td>Morioka, 1996³³</td>
<td>Cross-sectional (n = 282)</td>
<td>7-17 yrs</td>
<td>Firecrackers, noisy playthings, explosives</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>Exposure to impulse noises such as firecrackers, noisy playthings, and explosives may potentiate hearing loss in youth via a 4 kHz dip and/or high tone deafness.</td>
<td>Multivariate analysis, audiometry done in physician home</td>
</tr>
<tr>
<td>Kiukaanniemi, 1992³⁰</td>
<td>Prospective cohort (n = 39)</td>
<td>17-25 yrs (mean age 17.7 yrs)</td>
<td>Rifles</td>
<td>Audiometric threshold PTA</td>
<td>7-11 months</td>
<td>Mean hearing thresholds (0.5-20 kHz) were significantly different after approximately 1 year of military training, as compared to baseline. Within the conventional frequency range, only hearing threshold shifts in the right ear were statistically significant.</td>
<td>P-value of median threshold shift before vs after military service was calculated via sample t test</td>
</tr>
<tr>
<td>Kramer, 1982³¹</td>
<td>Retrospective cohort (n = 68)</td>
<td>11-18 yrs</td>
<td>Firearms use at least 4 times/month</td>
<td>Audiometric threshold PTA</td>
<td>N/A</td>
<td>Of 12 subjects found to have NIHL, 11 (92%) were regular gun users, suggesting a strong association between frequent gun use and hearing loss.</td>
<td>No statistical test of null hypothesis reported, post hoc P = .08 (exact test)</td>
</tr>
<tr>
<td>Segal, 2003³²</td>
<td>Retrospective review (n = 53)</td>
<td>4-14 yrs</td>
<td>Firecrackers, toy cap pistols</td>
<td>Audiometric threshold PTA</td>
<td>2-12 weeks, 6 months</td>
<td>Prevalence toward high frequency SNHL noted among youth ages 4-14 after seeking medical care for impulse noise exposure, which tended to persist at 6 months follow-up.</td>
<td>Most showed high frequency SNHL (notch at 4-6 kHz); statistical analysis not reported</td>
</tr>
<tr>
<td>Axelsson, 1993³³</td>
<td>Longitudinal follow-up study (n = 50)</td>
<td>5-12 yrs (approx)</td>
<td>Shooting pistols, air rifles, firecrackers, or fireworks</td>
<td>Audiometric threshold PTA</td>
<td>12 weeks</td>
<td>No significant correlation was observed between frequent shooting of pistols, rifles, playing with firecrackers or fireworks and elevated hearing thresholds.</td>
<td>Average frequency and intensity of sound exposures compared</td>
</tr>
</tbody>
</table>

Abbreviations: NHANES, National Health and Nutrition Examination Survey; NIHL, noise-induced hearing loss; NITS, noise-induced threshold shift; PIR, poverty-income ratio; PTA, pure tone average; SNHL, sensorineural hearing loss.
to audiometric criteria.\textsuperscript{22} Subjects 12 to 19 years old were found to have a significantly higher prevalence estimate of NITS than children 6 to 11 years of age, suggesting a possible role of environmental noise, but subjects were not specifically assessed for a defined exposure.

The third study (2010) used data from both NHANES III (1988-1994) and NHANES 2005-2006 and assessed the latter for an association between NITS and history of exposure to firearms or history of loud noise for >5 hours per week in adolescents.\textsuperscript{26} No significant association was demonstrated on multivariate analysis.

The fourth study (2011) utilized the same data sets and reiterated that there was no association between NITS and history of exposure to firearms or history of loud noise for >5 hours per week and that there was no association with headphone usage or loud noise exposure within the last 24 hours.\textsuperscript{19}

### Combined Noise Exposures

Multiple studies explored loud noise exposures from conglomerated or undefined sources (see Supplemental Table S1 appendix at www.otojournal.org).\textsuperscript{2,3,19,21,37,39,43,56-60} The largest study demonstrated a trend toward an association with hearing loss. Additional study results were mixed, and more studies reported no association between hearing loss and this more broadly defined exposure category,\textsuperscript{21,39,43,58-61} although sample sizes were frequently limited.

### Ear Protection

Four studies described the concurrent impact of ear protection on hearing loss in secondary analyses.\textsuperscript{19,21,38,42,43,62} One cluster-randomized control trial showed that participation in a hearing conservation program resulted in significantly increased usage of ear protection but did not demonstrate reduced levels of noise-induced hearing loss at the 3-year follow-up in adolescents exposed to farm noise.\textsuperscript{62} In another study of farm youth, ear protection was nearly significantly associated with reduced odds of noise-induced hearing loss (OR, 0.61; 95% CI, 0.34-1.09).\textsuperscript{42} Meanwhile, a longitudinal follow-up study demonstrated similar hearing protection rates among subgroups with large and small hearing threshold shifts.\textsuperscript{21} A cohort study specified that their occupational noise exposure variable was considered positive only if no ear protection was utilized.\textsuperscript{38}

Two studies investigated whether certain subgroups of children were more likely to utilize ear protection. One cross-sectional study showed that youths exposed to firearms were more likely to use hearing protection regularly. Females were significantly less likely to use hearing protection but also had significantly less noise exposure.\textsuperscript{19} A cross-sectional study indicated that among study participants who reported noisy educational (training for a future noisy job, e.g., car mechanic) or professional environments, approximately 40% reported seldom or never using hearing protection.\textsuperscript{43}
Discussion

Tobacco and noise exposure are personally modifiable risk factors that have a demonstrated negative impact on hearing loss in the pediatric population.

Tobacco Smoke

Nearly all relevant data suggest that even low levels of passive tobacco exposure, both in utero and obtained as secondhand smoke in childhood, pose a risk for pediatric SNHL.9,30-32 Thus, reduction without cessation appears insufficient to eliminate tobacco-associated risk both during pregnancy and afterward. Pregnant women may be advised that tobacco exposure as minimal as 1 to 4 cigarettes per day is sufficient to increase the risk of bearing a child affected by hearing loss. Parents may be counseled that there is a nearly twofold increase in hearing loss among adolescents exposed to secondhand smoke. Overall, the target with regard to prevention of hearing loss is thus ideally set at complete cessation, based on the currently available data.

There are limitations in these tobacco-related studies. The 4 analyses that assess maternal smoking all rely on self-report, subjecting these data to recall bias. Also, passive maternal exposure to tobacco smoke could not be assessed, and mothers who reported not smoking during pregnancy might have had secondhand smoke exposure, potentially altering results in the control group. In addition, an assessment of serum cotinine levels to determine secondhand smoke exposure was limited to a measurement in a single moment in time; the duration of secondhand smoke exposure was not known.

It has been suggested that tobacco exposure may negatively affect inner ear function via microvascular compromise and induced hypoxemia, among other possible causative mechanisms.63-65 In addition, secondhand tobacco smoke exposure in childhood or prenatal tobacco exposure have collectively been linked to many adverse health consequences,9,31,66-69 such as sudden infant death syndrome65 and behavioral/cognitive problems.70 Thus, beyond mitigating the associated risk to hearing, there are multiple benefits of complete cessation.

Noise

The most specific noise data suggest that we may counsel families that the following threshold exposures have been significantly associated with an increased risk of hearing loss in youth: (1) more than 4 hours per week or more than 5 years of personal headphone use, (2) more than 4 visits per month to a discotheque, (3) residing on a mechanized farm, and possibly (4) 1 year of military service. In addition, even single exposures to impulse noises such as firearms and firecrackers may result in audiological deficits. Data regarding in utero exposure and aircraft noise are mixed.

There are limitations in these noise-related data. The vast majority of studies determined exposure via patient/family questionnaire, with attendant potential for recall bias. Also, noise-induced hearing loss was defined inconsistently among studies, with varying frequency and threshold values of concern. In addition, the majority of studies did not report on the use of hearing protection, which is a potential key confounder.

Loud noise has the capacity to damage the cochlea,22,52,59 which may occur in ways that are not immediately detectable with clinical audiology. Mammalian studies have shown that even when reversible hearing loss occurs, there is residual loss of afferent nerve terminals and delayed degeneration of nerve after intense noise exposure.71,72 In addition, early-age noise exposure, while not necessarily resulting in threshold shifts, potentiated subsequent inner ear damage in an animal model.72 Thus, real but subclinical damage may occur, potentially offering a mechanism for some of the mixed results within this systematic review.

There may also be additional factors that are effect modifiers of the impact of noise on hearing. Certain subgroups may thus be substantially affected by noise, while others’ hearing remains normal even after identical exposures. Biassoni et al21 reported that some children had “tender” ears that were “easily damaged” by noise that would cause no change in children with “tough” ears, demonstrating significant differences between groups. While other studies did not mathematically evaluate this concept, some reported suggestive data. Two additional studies showed significant hearing losses in small subgroups of exposed children: One case series highlighted 5 pediatric patients who developed pure tone thresholds of 55 to 70 dB at 4 kHz.59 Another reported that a “few” children working with farm equipment had a “substantial decline.”62 When an effect modifier is present in a small subgroup, its impact may be diluted when evaluating a larger study population, which may also account for some of the mixed results in this data set.

The data regarding hearing protection are both limited and mixed. According to 1 report, nearly 99% of adolescents do not use hearing protection when exposed to loud music.73 A recent prospective pilot prevention program for children demonstrated that multiple-segment, interactive interventions resulted in sustainable changes in listening behavior.74 Furthering our understanding of such preventive strategies may ultimately improve our ability to counsel families toward better hearing health.

Potential Interaction between Tobacco and Noise

While these pediatric data have not focused on whether tobacco smoke and noise act synergistically to potentiate hearing loss, adult studies have suggested that concomitant exposure may increase risk. A cross-sectional study of adult metal workers suggested that those with tobacco and noise exposure had hearing loss exceeding a simple additive effect.75 Another cross-sectional adult study suggested a trend toward worse hearing in smokers with occupational noise exposure (OR, 1.85, 95% CI, 1.33-2.57) than in smokers without noise exposure (OR, 1.53, 95% CI, 1.03-2.29).76 Additional studies found that noisy work environments significantly increased risk if
subjects also smoked. Interaction as potential effect modifiers in multivariate models has been studied with a significant association in a cross-sectional study of Japanese metal workers but not among an observational incidence cohort of Australian adults. If a synergistic interactive effect exists in the pediatric population, then clubs and other environments where both may be present in a closed space would be of increased concern.

Conclusions

Specific threshold analyses of personally modifiable risk factors are limited. Future studies may focus on the impact of specific levels of exposure in order to provide actionable preventive guidance for children and families. Such preventive measures may ultimately preserve more audiological health than current interventions. Identification of susceptible subgroups may also guide future screening programs.

Acknowledgments

JJS would like to thank Thomas Y. Lin and the C.H.E.E.R. Network for support during the preparation of this manuscript. Stephanie Colello is also thanked for assistance with computerized and manual search corroboration.

Author Contributions

Adam P. Vasconcellos, acquisition of data, analysis and interpretation of data, drafting and editing manuscript, final approval; Meghann E. Kyle, acquisition of data, analysis and interpretation of data, drafting of tabular results, final approval; Sapideh Gilani, acquisition of data, analysis and interpretation of data, contributing to manuscript, final approval; Jennifer J. Shin, analysis and interpretation of data, acquisition of data, drafting and editing manuscript, final approval.

Disclosures


Sponsorships: None.

Funding source: None.

Supplemental Material

Additional supporting information may be found at www.otojournal.org/supplemental

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